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CONGENITAL TUBERCULOSIS—A SURVEY.

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GENUINE cases of congenital tuberculosis in human beings are undoubtedly very rare, and it has been generally thought that they only occurred when the mother was in an advanced stage of tuberculosis (probably suffering from miliary dissemination and "tuberculous bacillæmia"), but more recent observations (Weller, Warthin, Novak and Ranzel) seem to show that placental tuberculosis may likewise occasionally develop when the mother's tuberculosis is not far advanced, or even latent or quiescent.

I shall not discuss the observations and writings of Baumgarten, Maffucci, Jani, Walther, Jäckh, Karlinski, Wahlen, F. F. Friedmann, and others, regarding the possible primary infection of the human ovum from the mother or its infection by means of tubercle bacilli reaching it with the father's semen (at the time of impregnation or later on). Sitzenfrey (1909) sums up his conclusions on the available data regarding part of the subject in the following words: "If we desire to express concisely the result of our discussion regarding the transmission of the tubercle bacillus from the father or the mother to the germ, we may say that up to the present time no certain proof has been forthcoming that a transmission of this kind (in the human subject) ever occurs; but we must also say that we have no conclusive reasons for altogether denying the possibility of such an

occurrence.” According to S. A. Knopf (1902) “the extreme rarity of *primary* genital tuberculosis in vagina or uterus seems the best clinical evidence that direct paternal bacillary transmission of tuberculosis practically does not exist.” According to Lartigau (London Congress, 1901) there were four reported cases, but infection subsequent to conception was possible. On the other hand there were a good many indisputable cases on record of the maternal transmission of tubercle bacilli to the foetus.

The subject now to be considered is that of congenital tuberculosis due to intra-uterine infection of the child from the mother by way of the placenta and foetal membranes—with or without the presence of obvious macroscopic or microscopic tuberculous changes in the placenta. Some cases of placental tuberculosis without definite tuberculous infection of the foetus will also be referred to.

The examination of the placenta sometimes requires the expenditure of a very great deal of time and trouble before the presence of tuberculous lesions or tubercle bacilli can be detected in it. Mere macroscopic inspection can seldom be relied on. Indeed, Schmorl declared that as many as 20,000 sections might have to be examined under the microscope before tuberculous lesions, even when really present, could be found in a placenta. For demonstrating the presence of tubercle bacilli in the placenta Uhlenhuth's antiformin method has sometimes yielded excellent results and saved a great deal of time and (possibly fruitless) labour.

There are probably different ways in which the tubercle bacilli can pass from the mother to the foetus, and in the following pages various possibilities will be referred to. Very interesting are the cases of *late* congenital tuberculosis in which the infection of the foetus is supposed to have occurred during labour (*intra-partum*); the tearings and squeezings of the placenta, connected with the uterine contractions (labour “pains”) or artificial manipulations, may involve tuberculous foci and thus give rise to the transference of tubercle bacilli from the maternal placenta, to the foetal blood stream, more or less in the way suggested by Gärtner, Rietschel, Sitzenfrey, Pankow, Dietrich, and others. In cases apparently of this kind the child has lived for a considerable time, up to three or even six months.

To a certain extent such cases remind one of what is supposed to happen in some kinds of “traumatic tuberculosis,” that is to say, in cases where a contusional trauma or a wrench damages or ruptures an old quiescent tuberculous focus and thus leads to fresh local activity and possibly to metastasis or wide-spread dissemination of

the tuberculosis. Indeed, in tuberculous women childbirth and manipulations connected with it may act as a contusional trauma, or wrench, and in rare cases cause tuberculous infection of the foetus or (more probably perhaps) may give rise to metastasis or generalised tuberculous dissemination (acute miliary tuberculosis, especially involving the lungs) in the mother as the result of injury involving some (possibly previously quiescent) tuberculous focus in her pelvic viscera.*

Rindfleisch (1890) reported the case of a child, born in the ninth month of pregnancy, who died eight days after birth. The necropsy showed tuberculous caseous pneumonia and large caseous nodules in the liver.

Schmorl and Birch-Hirschfeld (1891) described the first† undoubted case of congenital (foetal) tuberculosis, in which it was evident that the tubercle bacilli reached the foetus by way of the placenta. A maid-servant, aged 23 years, died in the seventh month of her first pregnancy. The cause of her death was general miliary tuberculosis, probably a dissemination from primary renal tuberculosis. The placenta at first sight appeared to be almost normal, and microscopically showed no definite tuberculous changes, but it was found to contain tubercle bacilli, which were likewise present in blood-films taken from the umbilical vein. Slight microscopic changes and tubercle bacilli were detected in the liver, but not in other organs of the foetus. The authors suggested that the tuberculous infection of the foetus did not occur till shortly before its death, which apparently took place almost simultaneously with that of the mother. The foetus was known to be living 20 minutes before the mother died, but was found to be dead when delivered by Cæsarean section directly after the death of the latter.

Sabouraud's case (1891) was that of a child who died eleven days after birth. The mother died of pulmonary tuberculosis. The necropsy on the child showed miliary, and larger, tuberculous nodules in the liver and spleen, containing tubercle bacilli. Unfortunately the placenta was not carefully examined.

* Cf. F. Parkes Weber, 'Traumatic Pneumonia and Traumatic Tuberculosis,' London (Adlard), 1916, p. 31: references to cases of acute miliary tuberculosis following childbirth or abortion.

† That is to say, the first proved case in *human beings*. In regard to earlier undoubted cases of *congenital tuberculosis in cattle* see Johne's case of tuberculosis in a foetal calf at the eighth month of intra-uterine life (1885). The mother-cow had advanced pulmonary tuberculosis.

Thiercelin and Londe (1893) examined the body of a child who died four days after birth. They found tubercle bacilli, but no macroscopic changes, in the spleen, liver and kidneys. A guinea-pig which was inoculated with 2 c.c. of blood, taken from the umbilical vein at the child's birth, died of general tuberculosis. The child's mother had fatal pulmonary and intestinal tuberculosis. The condition in which tubercle bacilli but no actual tuberculous lesions are found in the fœtus or child has been named by Honl (1894) "*status bacillaris*" to distinguish it from true congenital tuberculosis with structural tuberculous changes.

Londe (1893), investigating the offspring of tuberculous mothers, gave his results of inoculation of guinea-pigs in six cases. One mother had pulmonary tuberculosis at an early stage and gave birth to a living child. Inoculation of a guinea-pig with the placenta gave a negative result. Four other women (with more advanced pulmonary tuberculosis) gave birth to children who lived only a short time. One of these four cases was the one recorded (1893) by Thiercelin and Londe (see back). In two of the other three cases inoculation of guinea-pigs with the placenta produced tuberculosis. In the remaining case of the four the inoculation test gave a negative result. The sixth case was that of a woman suffering from pulmonary, intestinal, peritoneal, and laryngeal tuberculosis, who aborted. Inoculations with the placenta and fœtal blood and organs all produced tuberculosis in guinea-pigs, most virulent when from the placenta. In no case was any macroscopic tuberculosis found in the fœtus or children examined (*cf.* remarks in previous paragraph).

Lehmann (1893) described a case in which there were definitely tuberculous changes in the placenta without any definitely tuberculous changes in the fœtus. A woman, aged 26 years, died from miliary tuberculosis in the eighth month of her pregnancy. A few minutes after her death the fœtus was removed by Cæsarean section, but it was (apparently just) dead. Its liver, on microscopical examination, showed small focal collections of round cells, but they were not associated with the presence of tubercle bacilli, and nothing suggestive of tuberculosis was discovered elsewhere in the fœtus. The placenta on the other hand contained miliary nodules which on microscopical examination were found to be of typical tuberculous structure and to contain a few tubercle bacilli.

In a second case (published in 1894) Lehmann found large tuberculous foci in the placenta. The mother suffered from chronic pulmonary and laryngeal tuberculosis. No tuberculous changes

could be found in the child, who died ten days after birth, but Lehmann thinks that from the foetal portion of the placenta the foetus itself may be infected though no definite tuberculous changes need manifest themselves in the liver and other organs during the early days of extra-uterine life (*cf.* further on, suggestions of Rietschel, Sitzenfrey, etc.).

In a third case (1894) of Lehmann there was certainly foetal tuberculosis; the placenta was however unfortunately not examined. The mother died with tuberculous meningitis three days after delivery. The (somewhat premature) child died on the day after birth, and the post-mortem examination showed miliary tubercles in the liver, spleen, one kidney, and myocardium, and also tuberculosis of bronchial and abdominal lymphatic glands.

Schmorl and Kockel (1894) described five cases of tuberculosis of the human placenta, in two of which tubercle bacilli were found also in the foetal liver. In two of the cases no evidence of tuberculous infection of the foetus could be discovered. In the fifth case the child died a few days after birth, but no post-mortem examination was made. All five mothers died before or not long (not many days) after delivery, and all of them had advanced or miliary tuberculosis.

In regard to three of their cases I will give further details from an abstract I made for a medical journal at the time: (1) A woman, aged 26 years, in whom a necropsy revealed acute general miliary tuberculosis; she was in the eighth month of pregnancy, and was brought to the hospital in an unconscious condition. Cæsarean section was performed during the agony of death, but the child died after two hours. (2) A woman, aged 25 years, who died with acute miliary tuberculosis during pregnancy. (3) A pregnant woman, aged 33 years, with chronic laryngeal and pulmonary tuberculosis, who died suddenly of profuse hæmoptysis. Cæsarean section was performed soon after the mother's death, but life was already extinct in the child.

In all three cases placental tuberculosis was found, but only very little in No. 3, and less in the placenta than in other organs in the other two cases. The tubercle bacilli were probably in all three cases carried to the placenta in the circulating blood; but in No. 2, where there was also tuberculous peritonitis, the bacilli, though less probably, might also have been carried from the peritoneum into the uterus by the Fallopian tubes. In cases of acute miliary tuberculosis the bacilli circulating in the blood must be equally distributed to all organs, one would think, but in order to explain why in Cases

1 and 2 less tuberculosis was found in the placenta than in the other maternal viscera it might be suggested that the placenta was more resistant to the tubercle bacillus. In all three cases tubercle bacilli were found in the foetal placental villi, but only in Case 2 were any bacilli found in the body of the foetus; even in that case there were no macroscopic changes, but bacilli could be detected by the microscope in the liver, and a lymphatic gland in the neighbourhood of the liver ("status bacillaris," see below). The authors thought that the reason why only few bacilli had passed into the foetal circulation was to be found in the changes produced in the vessels of the tuberculous villi of the placenta. These changes, consisting in the closure of vessels by the formation of hyaline thrombi and the overgrowth of the endothelium, to some extent seemed to constitute a barrier hindering the spread of tuberculosis and the entry of tubercle bacilli into the foetal circulation.

In regard to Case 2 of Schmorl and Kockel it may be noted that J. Honl (1894) named the condition in which tubercle bacilli could be found in the foetal organs, though unaccompanied by macroscopic or microscopic tuberculous changes, *Status bacillaris*, in order to distinguish it from the condition of true foetal tuberculosis, in which macroscopic or microscopic tuberculous lesions were present.

Kockel and Lungwitz (1894) investigated placental tuberculosis in cows in relation to foetal tuberculosis. I shall again quote an abstract which I made of their paper at the time. They carefully examined the uterine contents in two tuberculous cows killed during pregnancy. The foetus in the first case was of about the sixth month, and both macroscopically and microscopically gave evidence of tuberculosis in the liver, and in the portal and bronchial lymphatic glands. The mediastinal and some other lymphatic glands were also tuberculous, but were not examined microscopically. Some scattered grey specks were seen in the lungs just beneath the pleura, but no tubercle bacilli or typical tuberculous structure could be detected in them. No evidence of tuberculosis was found in the other organs.

The second foetus was about $4\frac{1}{2}$ months old, and evidence of tuberculosis was found in the liver, spleen, left kidney, left lung, and especially in the portal, mediastinal, one bronchial, and some other lymphatic glands.

In both cases there was extensive tuberculosis of the uterine mucous lining, and in the first case tubercle bacilli were found also (though few only) in the chorionic (foetal) villi; in the second case, however, no bacilli could be found in the tissue or vessels of the

chorion, though a considerable number of bacilli were seen in the epithelium, and occasionally also between the epithelium and the membrana propria of the chorion. In the epithelial cells containing tubercle bacilli the nucleus could often be seen properly stained, and this, the authors suggested, might lend support to the view that the epithelium offered a barrier to the passage of the tubercle bacilli from the mother to the foetus. It might likewise be suspected that the gelatinous tissue of the foetal villi and chorion did not afford a good nutrient medium to the development of the bacilli.

The authors thought that their observations supported the view of Birch-Hirschfeld, that the mode in which the tubercle bacilli passed from the maternal to the foetal circulation was by "growing through" the placenta, a view suggested to him by analogy from his observations in cases of anthrax. The authors further called attention to the localisation of the changes in tuberculosis of the foetus, namely, chiefly in the liver and the lymphatic glands, especially the portal, bronchial, and mediastinal glands.

Perhaps congenital tuberculosis is less rare in cattle than in human beings, but the Danish investigator, Bang, in a pamphlet translated by M. Bray, and quoted by Niven, stated that all the young calves in a herd of about 208 cows were injected (1892-1895) with tuberculin, and in the whole series only two calves were found to be tuberculous from birth, and those merely with localised insignificant tuberculous lesions. On the other hand, A. M. Bergmann, of Malmö (1910), found that, though congenital tuberculosis in calves was undoubtedly rare, it was not rare enough to be without practical importance. MacFadyean, in 1899, when he demonstrated a specimen of congenital tuberculosis in a calf at the Pathological Society of London, said that he had himself seen only one example before 1897, but had since then had three specimens forwarded to him. One of them was a calf, only a day or two old, showing tubercles distributed through the liver, spleen, lungs, and myocardium, many of the lesions being already calcified. (MacFadyean incidentally pointed out that calcification was not necessarily a criterion of the age of a tubercle.)

Bar and Rénon (1895) employed the guinea-pig inoculation test in order to obtain further evidence regarding the transmission of tuberculosis through the placenta. They selected five parturient women, who were tuberculous, and at each childbirth, when the umbilical cord was divided, they injected placental blood subcutaneously into guinea-pigs. The result was negative in three cases and positive in two. In the first of the two latter the foetus

had died shortly before birth, and though no definite tuberculous lesions were detected in its organs, two out of three guinea-pigs, inoculated with pulp from its viscera, succumbed as the result to tuberculous infection. The second child died of broncho-pneumonia on the fortieth day after birth. Both the mothers had advanced pulmonary tuberculosis and died soon after confinement.

Dolérís (1898) reported two cases of tuberculous mothers. In one of them, in whom the disease was limited to the lungs, the foetus apparently escaped. In the other no tuberculous lesions could be found in the foetal organs, but the guinea-pig inoculation test gave a positive result.

Auché and Chambrelent (1899) recorded the case of a woman, aged 40 years, with advanced tuberculosis of the lungs, etc., who died 5 days after spontaneous premature labour, in the seventh month of pregnancy. The infant (a female) was kept alive for 26 days in an incubator, though gradually losing weight all the time. The necropsy on the child showed advanced tuberculosis of the liver and spleen with less marked tuberculous changes in the kidneys and lungs. Tubercle bacilli were present in great numbers in the liver and spleen. There was also tuberculous endocarditis in the right ventricle; and the aortic and bronchial lymphatic glands, and those at the hilum of the liver were tuberculous. The placenta in this case showed tuberculous changes and contained tubercle bacilli. Animal inoculation tests with the child's organs, with pieces of the placenta, and with blood from the umbilical cord, all gave positive results for tubercle bacilli. This case showed that the foetal tissues, when once infected (by way of the placenta), do not form a bad soil for the growth of tubercle bacilli, as P. von Baumgarten supposed they did when he formulated his theory of congenital "latent tuberculosis."

G. d'Arrigo, of Naples (1900), made a series of experiments with guinea-pigs in order to study the transmission of tuberculosis from the mother through the placenta to the foetus. A certain number of female guinea-pigs were infected with tuberculosis and then impregnated by healthy males. Others were first impregnated and then infected. The latter nearly all aborted, but in the case of one which did not, tuberculous lesions were found in the placenta and in the foetal liver. Those rendered tuberculous before impregnation showed varying lesions (according to the period at which they were killed) in the placenta, chorion, etc., and foetal liver. The animals born at full term were feeble and died in 5 to 16 days from general tuberculosis.

Bossi (1903) described a series of experiments on gravid rabbits and guinea-pigs with reference to the transmission of tubercle bacilli from mother to foetus. When it was known that the animal was pregnant tubercle bacilli were given by intravenous, subcutaneous, or intra-peritoneal injection. The results were watched, and in no single instance were tubercle bacilli found in the viscera or blood of the foetus. In only three cases were bacilli found in the placenta, and in each of these three cases the animal aborted. Bacilli were never found in the milk. Abortion was a frequent result of the injections, and all the mother animals died in the puerperium, at various periods after the labour. In these experiments the conditions were obviously very unlike any that occur in Nature.

E. Runge (1903), a pupil of Schmorl, made a careful study of a case of placental tuberculosis in a woman, aged 32 years, who died with old pulmonary tuberculosis and recent disseminated miliary tuberculosis, in the fourth month of pregnancy. Hæmorrhage between the placenta and the uterine wall had led to partial separation of the placenta. Runge found foci of round cells, containing tubercle bacilli, in the decidua basalis. These tuberculous foci showed necrotic changes in their centres and had led to destruction of the walls of neighbouring blood-vessels, thus giving rise to the above-mentioned hæmorrhagic partial separation of the placenta. Unfortunately the foetus was not examined.

Stoeckel, at Erlangen (1903), exhibited the abdominal organs of of a child (of a tuberculous mother), showing congenital miliary tuberculosis. Microscopic examination of tubercles in the liver proved the presence of numerous tubercle bacilli. The child died 14 days after birth, and the stage of the tuberculosis was certainly too advanced for the idea of a so-called *intra-partum* infection of the foetus to be entertained.

Simmonds, of Hamburg (1904), related the case of a woman, aged 31 years, who died with tuberculous peritonitis 20 days after having given birth to an apparently healthy child at full term. At the necropsy the tuberculous peritonitis seemed to have started from tuberculous endometritis and salpingitis. The child gradually wasted, and died two months after birth. On post-mortem examination it was found to have advanced pulmonary tuberculosis with extensive caseous changes. There were tubercles in the pleuræ and in the liver. The bronchial, but not the mesenteric and abdominal, lymphatic glands were affected. Owing to the advanced stage of the disease Simmonds thought it must be of congenital (intra-uterine) origin and connected with the tuberculous endometritis of

the mother, but unfortunately the placenta was not thoroughly examined. This may have been an example of *late (intra-partum)* congenital tuberculosis (see further on).

Schmorl and Geipel (1904) contributed a valuable paper on tuberculosis of the human placenta. They examined twenty placentas from tuberculous women, and in nine of them discovered tuberculous foci. Of the 20 women one died of miliary tuberculosis, and one of tuberculous meningitis following tuberculosis of the bronchial glands; eleven died with advanced tuberculosis during or soon after the puerperal period; four suffered from moderate and three from commencing pulmonary tuberculosis. Of the 20 placentas 18 were of the terminal period of pregnancy, one was of the seventh, and one of the eighth month. Evidence of tuberculosis was discovered in eight of the full-term placentas and in the seventh month placenta (in nine out of the twenty, as stated above). The authors described exactly the various tuberculous changes which they found in the placentas and classified them into four groups. In one placenta, that from the woman with miliary tuberculosis, the changes were so advanced that they could be recognised with the naked eye as certainly tuberculous in nature. The authors thought that when the placenta was tuberculous, the foetus, if not already tuberculous, might be infected from the placenta at birth (*cf.* suggestions of Rietschel, Sitzenfrey, etc.). In such cases the newly born child would at first show no tuberculous lesions (that is to say, there would be no evidence of tuberculosis), though the tubercle bacilli in its organs might give rise to anatomical tuberculous changes later on.

Martha Wollstein (1905) described a case of congenital tuberculosis. The mother died six days after confinement and the child died 19 days after birth. The placenta showed advanced tuberculous changes and the infant showed miliary tuberculosis of the lungs, spleen, liver, kidneys, and mesentery.

F. Hamburger (1906) described a case of apparently congenital tuberculosis in a child who died at the age of 7 weeks. The mother had died of tuberculosis soon after the child's birth. The necropsy on the child showed generalised subacute tuberculosis, with chronic caseous tuberculosis of the lymphatic glands at the hilus of the liver. This may have been an example of *late (intra-partum)* congenital tuberculosis (see further on).

Jung (1906) reported the case of a tuberculous woman, who died thirteen days after spontaneous delivery of a child in the eighth month of pregnancy. The necropsy showed renal, osseous, peri-

toneal, and intestinal tuberculosis. There was likewise tuberculous endometritis. The left Fallopian tube was full of caseous material; in the right tube there was commencing tuberculosis in the portion furthest removed from the uterus. In the chorion close to the edge of the placenta there were many miliary tuberculous nodules containing tubercle bacilli. The child was taken away by the father, and its ultimate fate is not stated.

W. Carl (1907) found extensive tuberculous changes in the placenta, together with tuberculous caseous endometritis, in the case of a woman, aged 25 years, who died from tuberculous meningitis in the seventh month of pregnancy. By microscopic examination of the placenta Carl discovered a condition of "endometritis deciduæ basalis caseosa," which had already spread to the foetal villi. In the organs of the foetus itself neither tuberculous changes nor tubercle bacilli were detected.

Herman (Royal Academy of Medicine in Belgium, 1908), rendered a large number of guinea-pigs tuberculous by feeding them with human tubercle bacilli. During the period of the experiment the infected guinea-pigs gave birth to 47 young ones (including one foetus), and in two of the 47 Herman discovered tuberculous lesions in the liver, containing tubercle bacilli, proving the presence of congenital tuberculosis.

Huguenin (1908) examined a foetus born prematurely of a severely tuberculous mother, and was able to prove the presence of tubercle bacilli in the foetal organs by the guinea-pig inoculation test. In another (similar) case the guinea-pig had not yet been killed, but a nodule had already formed at the site of inoculation.

Honjio (1909) described a case of human congenital tuberculosis in a five-months foetus. The retroperitoneal lymphatic glands were enlarged and were proved to be tuberculous, both by microscopical examination and by the animal-inoculation test. The mother died of pulmonary and intestinal tuberculosis with general miliary dissemination.

Rietschel's case (1909) was that of the child of a woman suffering from advanced tuberculosis, who died one day after childbirth. The infant, as soon as born, was removed from its mother, but in about a month's time presented clinical signs of tuberculosis and died after three months (from generalised tuberculosis). Rietschel argued strongly in favour of the tuberculosis in this child having been congenital, and not acquired during extra-uterine life. Unfortunately no microscopical examination of the placenta was made, but in one of two somewhat similar cases (Sitzenfrey) the placenta was

examined and proved to be tuberculous. Rietschel suggested that in such cases the child might have escaped infection with tubercle bacilli during intra-uterine life until the actual onset of the uterine contractions of labour. With A. Gärtner (1893) he explained the likelihood of a tuberculous lesion in the placenta being ruptured during some stage of the labour, so as to cause tubercle bacilli to be discharged from the placenta into the foetal circulation. This *late (intra-partum)* kind of congenital infection might, it was maintained, explain why the signs of tuberculosis sometimes developed such a relatively long time after birth, as to lead to the supposition that the disease was of extra-uterine origin. In one case supposed to be of this kind (Sitzenfrey) the child lived to the age of six months.

(To be continued).

THE TEETH IN RICKETS.*

By J. LAWSON DICK, M.D., F.R.C.S.

THESE observations on the teeth in rickets form part of a general examination of 1000 school children attending L.C.C. schools in the East End of London. Without entering into the larger results it may be stated that 80 per cent. showed distinct evidence of rickets. In other words, practically all children living under conditions such as prevail in the East End of London have to struggle through a rickety phase of their existence in the first two years of their life, and this struggle leaves its marks in certain definite signs or stigmata which persist during the whole of the child's school career. A special point was that the children were mostly Jewish children. They were purposely taken to show the relationship between nutrition and rickets. As a rule the nutrition, especially the fat nutrition, of Jewish children is very good, and over 80 per cent. of the children were breast fed for from twelve to eighteen months.

Defect in the calcium metabolism, especially in the laying down of calcium salts throughout the skeleton, is the most prominent feature in rickets, and the one responsible for most of the physical signs which mark the disease. Nowhere is this factor more marked than in the teeth. In rickets caries is exceedingly common, and hypoplasia, or defective calcification of the enamel, is well marked.

* A paper read before the Section of the Study of Disease in Children of the Royal Society of Medicine on May the 26th, 1916.

A hypoplastic condition of the teeth is characterised to the naked eye by a defective formation of the enamel and frequently stunted growth of the teeth. There may be only a pitting, producing a honeycombed appearance of the enamel, or the enamel covering is slight and the cutting edge of the teeth presents sharp points, giving a characteristic appearance to the tooth. The defect usually extends from the cutting edge and may, in severe cases, involve the whole crown. As in syphilis, the condition is found typically in the permanent dentition, but whereas notched incisors and the contracted first molars of syphilis are but rarely met with in school children, the hypoplastic teeth of rickets are common. In everyday life one constantly sees these teeth and notes how well they often last. Thus frequently a smoker is seen whose teeth have been worn down in depth, and only short stumpy teeth are left, with a layer of enamel all round the cutting edge and the dentine exposed in the centre of the biting edge.

The calcification of the teeth begins about the fifth month of intra-uterine life, and the following diagrams (Figs. 1, 2, 3) give the rate of progress of calcification at various periods for both the temporary and permanent teeth.

The only teeth of the permanent set which show any signs of calcification at birth are the cusps of the first molars. Fig. 3 shows the portion of the enamel which has undergone calcification at the end of the first two years of life, and the parts affected by the commonest form of hypoplasia. In the typical form of hypoplasia commonly met with, the teeth affected are the central and lateral incisors, the tips of the canines, and the crown of the first molars. The condition is symmetrical, affecting both jaws. Usually the depth of the defect is greater in the enamel of the central incisors than of the lateral incisors, and it will be noted that the enamel affected is identical with that laid during the first two years. The condition is almost pathognomonic of rickets. Rickets is the only condition which interferes with the deposition of calcium over this prolonged period.

In cases of acute illness, especially fevers, the finger-nails are apt to show grooves marking the state of depressed growth of the cells of the bed of the nail during the progress of the disease. In the case of the teeth a groove more or less broad, or even a succession of grooves with healthy enamel in between, may mark attacks of grave illness in the child. Frequently without a history, a shrewd guess can be made at the period of occurrence of some serious illness by the part of the enamel of the teeth which is affected.

But it is usually a grave and prolonged illness which thus leaves its mark, more especially measles followed by whooping-cough. Scarlet fever is less likely to have this effect. The internal secretory glands apparently play a large part in the defence of the body during attacks of infectious disease, and probably no glands play

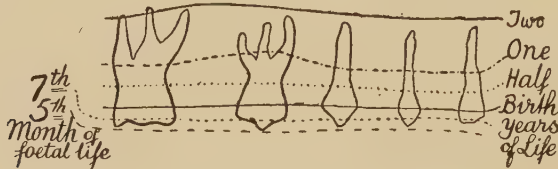


FIG. 1.

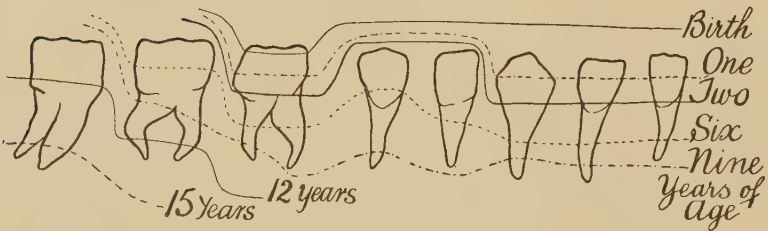


FIG. 2.

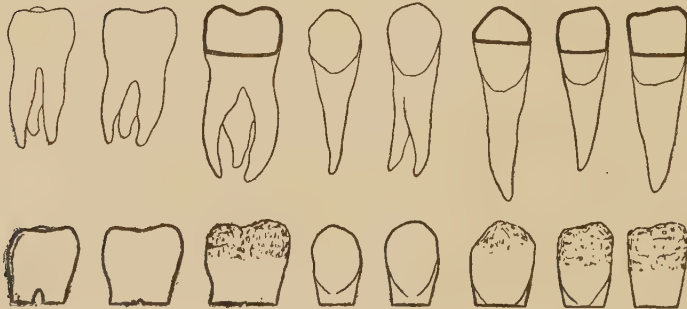


FIG. 3.

such a large part as the thyroid. The thyroid apparatus, more especially the parathyroid, as pointed out by Loeb, is largely concerned in governing the calcium metabolism of the body, and any depressing cause which makes extra demands on the glands over a prolonged period, when calcium is being actively assimilated and laid down in the bones and teeth, is liable to show itself in defects of the enamel.

Hypoplasia due to congenital syphilis is rare, whilst the form due to rickets is exceedingly common among school children. At birth calcification has involved the crowns of the deciduous incisors, the cusps of the deciduous canines and deciduous molars, and the tips of the cusps of the first permanent molars. The portions of the temporary enamel not yet calcified at birth in the temporary set frequently suffer from hypoplasia. Any disturbance in the function of the ameloblasts, the cells which, according to Tomes, either secrete the enamel or in which the calcium salts are deposited, will lead to the enamel being thin in parts and to the lime salts being imperfectly laid down, so that the compact enamel is more easily disintegrated. In other words, the agents which determine the tendency to decay are those which affect the soft enamel organ in the earliest history of the tooth and not those which affect the enamel after the tooth has been erupted. Ewan Waller holds that alteration in the quantity and quality of the saliva in hypothyroidism are prime factors in the causation of decay. Lactic acid produced by fermentation of carbohydrates is generally held to be the chief factor in producing caries. Waller believes that in thyroid inadequacy the parotid saliva is rendered less alkaline, or actively acid, from deficiency of calcium bicarbonate in the saliva, and that therefore the saliva fails to neutralise the lactic acid. Possibly this is a factor, but the chief factor is probably the manner in which the calcium salts are deposited in the soft enamel organ.

Seafaring communities are said to have good teeth, and this is generally attributed to the action of ozone and pure air. But more likely causes are the favourable conditions under which the children of such communities are brought up in early life, as regards fresh air, sunlight and ventilation, and the consequent better balance of the internal secretions controlling the processes of growth throughout the body. Professor Keith, in his fascinating book, 'The Antiquity of man' (p. 12), speaking of the Coldrum collection of Neolithic men, draws a comparison between Neolithic and modern man as regards their teeth :

"Amongst modern Kentish folk, as is the case all over modern Britain, there is a tendency to crowding and irregularities of the teeth; the palate and jaws do not grow and expand sufficiently in youth to give room for a symmetrical eruption of the teeth. The nose is narrow and the palate contracted, and its vault is high. The teeth are not worn down as in Neolithic men; they are very liable to be attacked by caries. The front teeth, when the jaws are closed, do not meet edge to edge as in primitive races; like the blades of

scissors, they overlap, the lower passing behind the upper. In the Neolithic people all these modern characters are absent. Abscesses or gum-boils at the roots of the deeply ground teeth were, however, common; but there is not a single carious tooth to be seen in the Coldrum collection. The teeth are regular in their arrangement, the palates were well formed, but in actual size the teeth possess the same dimensions as those of modern English people. All these changes, which are appearing in the teeth and jaws of modern British people, arise, we suppose, from the soft nature of our modern diet. We believe that were modern men to resume a Neolithic diet their teeth and palates would again be moulded in the ancient manner."

The difference is, however, hardly capable of such easy solution. Rickets was probably a disease unknown among Neolithic infants, as is largely the case among native races to-day, notwithstanding frequent privations as regards food. Contrast the life of Neolithic men living under primitive conditions, rising with the sun and going to rest at sunset, and living in small communities scattered over wide areas, with that of the slum conditions under which so large a proportion of our population lives to day. It is not a question of food, but of the deprivation of fresh air, exercise and sunlight, which profoundly alters the metabolism of the child and produces aberration in the growth not only of the bones and teeth, but in probably every tissue of the body. Natural selection is not always beneficial in its action. Nature is ever mindful of the continuity of the race, and if the slum-dweller is essential for its continuity an individual capable of adapting himself to such conditions will undoubtedly be produced.

In judging of the presence of hypoplasia, it was found practically impossible to make accurate observations on the temporary teeth of infants of school age. Caries was so universal and extensive that it completely masked the hypoplasia. It was evident that a hypoplastic condition of the teeth was common in the temporary set, and was, in all probability, the chief factor in bringing about premature decay. For the purposes of statistics only the records of the permanent dentition have been taken, and in marking a case as one of hypoplasia, only the severer forms are admitted, and cases of slight pitting, chalky-looking patches in the enamel, and so on, have been excluded, although microscopically such teeth would be found markedly defective and liable to disintegration. Of the 586 rickety cases in which a record of the permanent teeth could be taken, 42 per cent. had normal teeth and 58 per cent. had defective

teeth; 20 per cent. of these showed hypoplasia frequently combined with decay, and 38 per cent. had decayed teeth. This is not equivalent to saying that 42 per cent. of school children have normal teeth. As already pointed out, all infants with a record only of temporary teeth have been excluded, because these teeth were so universally decayed as to make accurate observations on the structure impossible. Again, most of the children with records of the permanent teeth were about the ages of 12 or 13 years, when all the permanent teeth have been erupted except the third molars, and caries has least time to make its appearance. It is a somewhat quaint commentary on the general state of the teeth of the community to have to explain why only 58 per cent. of the individuals are given as having defective teeth.

Of the cases with carious teeth, the lower first molar was decayed in 80 per cent., the upper first molar was decayed in 30 per cent., one or more lower premolars in 30 per cent., and one or more upper premolars in 12.5 per cent. The incisors, canines, and second molars were seldom decayed. The shape of the incisors and canines must protect them from many of the causes of decay to which the flattened grinding molars are subjected. The lower first molars decay out of all proportion to the others, and their earlier eruption is not a sufficient explanation of this. It is to be attributed rather to the main part of the enamel of the crown having been laid down in the first two years of life, when rickety conditions are operative. Naturally the lower teeth, which lie in the well of the mouth, will suffer more seriously from deleterious influences which surround the teeth.

Even though no macroscopic change indicative of hypoplasia is to be found in these teeth, in all probability their microscopic character is distinctly affected. One practical point may be noted. Frequently on looking into a child's mouth the two milk molars or the first permanent molar will be seen with blackened exposed dentine and the enamel on the surface of the crown removed. Closer examination will show that these are still effective teeth and that there is no pyorrhœa or gum irritation around them. The surface of the tooth can be freely touched with the spatula. The dentine of the tooth seems to have become consolidated, and usually, though the teeth do not look well, they can be safely left, the process of decay having become arrested. Frequently it is not a process of decay in the ordinary sense, for the thin defective enamel on the surface of the crowns of the grinding teeth can sometimes be seen breaking off in distinct flakes. Again, many of the conditions conducive to

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rickets are present in the parents of these children, and *a priori* it might be expected that this would frequently be a congenital defect. In such a case in a certain proportion of children the milk incisors should show hypoplastic changes. The enamel of the crown of the milk incisors begins to be laid down about the seventeenth week, half-way through intra-uterine life, and has progressed to a considerable extent at the time of birth. At birth, half the crowns of the incisors, the tips of the canines and the cusps of the molars are calcified in the temporary set, and by about six months after birth the calcification of the crowns is completed. Careful search for a hypoplastic condition of the milk incisors during a long period both in school children and in the babies who have attended the Hackney Mothers' Centre has failed to detect a case. It is strong presumptive evidence that rickets is not a congenital condition, and is another instance of the care that Nature takes that, whatever else suffers, the germ at least is protected during the intra-uterine life. If in achondroplasia the milk incisors showed signs of hypoplasia, this would be excellent evidence to link it up as a congenital form with rickets.

A later form of hypoplasia is every now and then seen in which the two premolars and the second molars are affected, whilst the incisors, canines and first molars are not affected. This latter form of hypoplasia must be due to influences acting on the child from the second to the sixth year. Association has been recognised between lamellar cataract and hypoplastic teeth. Mr. Norman Bennett collected twenty-two cases of lamellar cataract, all of which showed well-defined hypoplasia in the permanent teeth, and in thirteen cases there was a history of convulsions whilst teething.

TWO CASES OF SPLENECTOMY FOR BANTT'S DISEASE.

By W. F. CHOLMELEY, F.R.C.S.,

Hon. Surgeon General Hospital, Wolverhampton.

In the 'British Medical Journal' of March the 11th, 1916, p. 365, was published an account, with commentary, of a case of splenectomy for splenic anæmia in a woman, aged 45 years, in Addenbrooke's Hospital, Cambridge. Below is an account of two cases in the General Hospital, Wolverhampton, in children aged 6 and 12 years.

CASE 1.—A boy, aged 6 years, was seen by Dr. J. A. Codd on January the 12th, 1916. The history given was as follows: The

boy had been ailing for about three years, being pale and languid; his appetite had been good, but he was getting thinner. He had had pain in the epigastric region at times, but there had been no vomiting or diarrhoea. No history of hæmorrhages could be obtained. He had had a good deal of cough. On admission into hospital he was seen to be very pale, the skin had a lemon tint, and there was slight icteric tinge of the conjunctivæ. The spleen was very large, also the liver. There was considerable hæmaturia, although no history of it could be obtained. The temperature on admission was 102° F., but it fell to normal the next day, and did not rise above 99·4° F. during the whole of his stay in hospital, and was above normal on twelve days only. The sole medical treatment that he had was liq. arsenicalis $\mathfrak{m}3$, t.d.s., which was begun on January the 17th, and continued for a few days. I operated on January the 21st. The spleen, if at all different, was slightly smaller, but reached well beyond the edges of the rib cartilages. The abdomen was opened by a vertical incision above the umbilicus, and enlarged to the left by cutting transversely through the rectus just above the level of the umbilicus. A few adhesions were found, but were easily dealt with. The removal of the spleen gave no trouble, the pedicle being tied in two portions. There was hardly any bleeding. The wound was sutured in layers and a small tube drain left in for twenty-four hours. There was exceptionally little shock. The liver was much enlarged. The day after the operation there were well-marked evidences of improvement. There was a healthy pink colour in the lobes of the ears, the cheeks, lips, and conjunctivæ, the boy was very comfortable, and did not appear as if he had so recently gone through a severe operation. There was a slight amount of pyrexia for a few days, but the temperature became normal on the fifth day and remained so. Unfortunately no differential blood-count was taken, the number of red and white cells only being estimated.

January the 19th.—Two days before the operation, red cells, 1,850,000; white cells, 6500 per c.mm.

February the 7th.—Seventeen days after the operation, red cells, 2,700,000; white cells, 11,500.

He left the hospital on February the 14th. His blood was examined again on March the 9th, the result not being so good as on the last occasion. Red cells, 2,425,000; white cells, 15,600; hæmoglobin, 70 per cent. I saw him last on April the 27th; he was still rather pale and feeble, though not nearly so bad as before the operation. I have not been able to see him since.

CASE 2.—A boy, aged 12 years, was admitted into the hospital on February the 10th, 1916. The history given was as follows: He had apparently been quite well, except for some slight paleness, until a week before admission, when he had a bad fright on the occasion of a Zeppelin raid in his district. He complained of pain in the back, abdomen, and head. There had been no vomiting or hæmorrhages; there had been some polyuria. He had diphtheria when two years old, and was said to have been anæmic ever since. The notes written on admission were: Patient has no pain but in his head, very anæmic, no icterus. Heart not enlarged, marked pulsation and systolic bruit at apex and over pulmonary area. Lungs normal. Abdomen: Spleen much enlarged, notch marked and somewhat nodular on surface. Temperature, 102.6° F. I saw the boy on February the 19th. His condition was about the same. The blood-count was: Red cells, 1,330,000; white cells, 5000; hæmoglobin, 35 per cent. The report added: "Film shows slight degeneration of red cells in shape and staining properties." I hesitated about operating on this case on account of his heart, but finally decided to risk it. The spleen was removed on March the 3rd. I used the same kind of incision as in the former case. There were rather more adhesions, but no difficulty was experienced in getting at them or removing the spleen, which in this case was much larger. It weighed $2\frac{3}{4}$ lb. The patient stood the operation very well in spite of his heart trouble, and his recovery was rapid. Very little blood was lost at the operation. There was more pyrexia in this case before the operation, the temperature ranging between 97° and 102.8° F., but practically none after. The improvement was not so marked immediately after as far as the naked eye was concerned.

February the 29th.—Three days before operation a blood-count was taken. Red cells, 1,600,000; white cells, 11,000.

March the 11th, a week after, the blood-count was: Red cells, 2,900,000; white cells, 10,000; hæmoglobin, 70 cent.

He left the hospital on March the 19th, and on April the 26th the blood was again examined, with the following result: Red cells, 2,800,000; white cells, 53,000; hæmoglobin, 60 per cent. Differential count: Polymorphonuclears, 40 per cent.; lymphocytes (including many in degenerated and transitional stages), 53 per cent.; eosinophils, 1 per cent.; large hyaline, 5 per cent.; mast-cells, 1 per cent. I saw him last on April the 27th, when he seemed to be much improved, but I have not been able to see him since.

The incision used in both cases may seem to be unnecessarily large, but that is more than counterbalanced by the ease with which

deep adhesions can be reached and dealt with, and a large spleen delivered with the minimum amount of manipulation and pulling on the pedicle, both of which are, I am sure, the chief cause of shock in these operations. The increase in the length of the operation due to the making and suturing of a much more extensive wound is more than compensated for by the rapidity and ease with which the spleen can be removed and adhesions dealt with. If the rectus is sutured to its sheath by two parallel rows of stitches and divided between them, and the wound sewn up carefully in layers, there is not the slightest risk of subsequent hernia. The improvement in the first case was not nearly so good as in the second. This was due, I think, to the difference in age, the length of the previous ill-health, and the more feeble condition of the boy. I do not think that the history given in the second case can have been correct; the spleen, which reached nearly to the middle line, must have been obviously enlarged for some time. He had had some anæmia for some years before he was admitted, and no doubt the spleen had been enlarged but not noticed.

It is unfortunate that only one differential blood-count was taken, but there were many insuperable difficulties in the way. The only great difference in the blood-counts of the two cases is the number of white cells in the last examination of the second boy. It is early yet to form an opinion as to the future in these cases, but I feel very pessimistic about the younger one.

Note by Dr. Codd.

The first case presented the typical appearance of the lemon-yellow tint with slight icteric tinge of conjunctivæ. These symptoms, which always suggest pernicious anæmia in the adult, should always suggest Banti's disease in a child, and the first thing to do is to feel for the spleen, and the next to have a blood-count. Leukæmia does not show in children this lemon tint in the early stages. The appearance is more like that of the "large white face" as seen in "large white kidney."

The second case did not show this lemon tint. The history given was very dogmatic. The relatives stated that immediately after the bombs were dropped by a Zeppelin close to his house he became ill and his abdomen began to enlarge, this enlargement being due to the growing spleen. Although he was familiarly called by us the "Zeppelin spleen" case, I am inclined to consider that the shock-causation theory should be received with caution unless it were substantiated by other well-marked cases.

In a former case of Banti's disease that was under my care I first treated him with X rays and arsenic without any definite benefit. I then transferred him for splenectomy, and after this he made apparently a complete recovery. This failure with X rays is in striking contrast to the markedly beneficial influence of X rays in lymphadenoma and leukæmia, and I should never treat another case of Banti's disease with X rays, but transfer him at once for splenectomy, which acts like a charm. The huge blood-destroying organ is removed and excessive hæmatolysis stops at once.

Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Friday, May the 26th, 1916.

DR. E CAUTLEY, *Ex-President, in the Chair.*

Optic Neuritis (? Cerebral Tumour).—Dr. CAUTLEY.—A boy, aged 6 years 8 months, attended as an out-patient at the Belgrave Hospital for Children on April the 17th, under Dr. Prentice, who admitted him the following week with these notes:

"April the 10th: Headache for two weeks. Wakes up screaming at night. Anorexia, furred tongue, offensive breath, (?) vomiting, B.O.

"April the 17th: Not so well; headache, retching, drowsiness. Arm reflex greater on the left than right side; normal abdominal reflexes; knee-jerks equal and brisk. Gait unaffected. Optic discs swollen, and hæmorrhage on the left disc. He vomited during the afternoon but did not complain of headache."

Since being in hospital he had not suffered from headache or vomiting.

Mr. Bishop Harman's report on eyes (May the 9th): Bilateral papillitis, 1.5 D. elevation. Fine interstitial hæmorrhages at left disc, no exudation. No disorder of fixation."

There was no albuminuria. The gait was a little stiff.

Optic Neuritis (? Cerebral Tumour).—Dr. CAUTLEY also showed a girl, aged 11 years, admitted to Belgrave Hospital on April the 27th. One month before she had had two fits. Since then she had had almost constant headache and vomiting, the result of the headaches. On April the 26th she had another fit. She was a strong, high-coloured, healthy-looking girl. Her abdominal reflexes were practically absent; deep reflexes depressed, the left rather more marked than right; plantar reflexes absent. Her head was tilted somewhat towards the right shoulder, which was a little raised. Gait somewhat unsteady, (?) cerebellar, and she tended to fall backwards and to the left. Her left side was a little weaker than the

right and showed slight inco-ordination, probably sensory; and for the same reason the left movements were clumsy. The eyes showed nystagmoid movements, lateral and rotatory, and slight left convergent squint. Perhaps there was a little weakness of the left facial nerve, and there was double optic neuritis.

Mr. Bishop Harman's report (May the 9th): "Nystagmus on extreme duction, tendency to left convergent squint; well-marked papillitis in each eye, about 2 D. elevation, a trifle more on right side; no hæmorrhages or exudation; slight silkiness of right macular region."

Since being in hospital no special symptoms had been noted and she had not suffered from headache or vomiting.

Infantilism.—Dr. CAUTLEY.—Boy, aged 6 years 8 months, sixth child. He was said to have been a puny babe, but to have grown up to 3 months of age. At the age of 9 months he only weighed $4\frac{1}{2}$ lb., and from 13 to 18 months of age he remained stationary at 6 lb. 1 oz. He was entirely breast-fed for one and a half years and partially so for another six months. Proprietary foods were given up to the age of $4\frac{1}{2}$ years, and since then he had had ordinary diet. His mother suckled her previous baby for eighteen months, up to her confinement with this child. During the first year of life he was remarkably somnolent, and at the age of 3 years he had diphtheria. At times he was troubled with constipation, and occasionally with cramps in the hands and feet. He had had no such troubles during the two months he had been under observation.

Condition on May the 18th.—Height, $26\frac{1}{2}$ in.; weight, 16 lb. 14 oz.; head, 17 in. He was pot-bellied, markedly rachitic, and the liver had dropped well below the costal margin. There were enlarged tonsils, adenoids, and moderate adenitis. The skin was elastic and complexion rather dull. He spoke fairly clearly, and mentally appeared about 4 to 5 years of age. He had twenty good teeth, the first having been cut at the age of 2 years. His appetite is poor.

On April the 22nd he developed pertussis. This induced convulsions, once on April the 24th and four times on April the 25th, and was followed by general bronchitis. On admission on March the 25th he weighed 15 lb. 12 oz., and on April the 20th he had gained 28 oz. Since then the pertussis and bronchitis had caused loss of weight. The Wassermann reaction was negative.

There appeared to be no special cause for his backward development, beyond malnutrition and unsuitable feeding during early life. He had been taking polyglandin and improved, but the improvement might have been due to food and nursing.

Giantism.—Dr. W. MITCHELL SMITH.—Girl, aged 12 years 9 months.

Family history.—Patient was the seventh of ten pregnancies; two miscarriages; five died at or soon after birth; three living and well; brother, aged 20 years, height 5 ft. $9\frac{1}{2}$ in., weight 16 st.; a brother, aged 11 years, normal; and present case. Father suffered from diabetes; once 17 st., now 14 st. Mother healthy. Maternal grandmother reputed to be 32 st. at time of her death.

Present condition.—Height, 5 ft. $4\frac{1}{8}$ in.; weight, 17 st. 7 lb. Very obese, with equal distribution of fat; large framework, especially across the hips. Pubic hairs. Menstruating regularly since last Christmas. Blood-

pressure 125 mm. Hg.; urine normal; optic discs normal. Mentality normal for girl of her age (Standard VII); excellent memory, placid disposition, gross eater.

Skiagram of sella turcica showed that—(1) it was very well developed for a girl of her age; (2) there was no erosion of anterior or posterior part to indicate a tumour of any size.

Juvenile General Paralysis.—Dr. J. WALTER CARR.—Boy, aged 8 years. There was nothing in the family history to suggest syphilis, and the patient did not present any of the stigmata of the disease. He had not had any previous illness of importance. For about six months his walk had been getting increasingly jerky, and he had sometimes fallen down. For the last three months his pupils had been noticed to be unequal. Recently his speech had been getting indistinct and mumbling. Formerly he was rather bright at school, but of late had ceased to make any progress; had been unable to write, and had lost his memory. He had not had any convulsions. He was a healthy-looking, rather stout boy. He showed marked mental impairment, with distinct delusions of exaltation—for instance, he talked of having knocked down a policeman and killed two Germans. He had incontinence of urine and often of feces also. The left pupil was larger than the right and reacted very sluggishly to light; the right pupil reacted normally. The left optic disk was very considerably paler than the right. He had a distinctly spastic gait, with double ankle clonus and exaggerated knee-jerks. The triceps and supinator jerks were also increased. It was not possible to be sure whether the plantar reflexes were flexor or extensor. There was no ataxy. Wassermann reaction of blood positive.

Hemi-hypertrophy — partly Crossed.—Dr. A. S. BLUNDELL BANKART.—Boy, aged 6 years. Asymmetry of face and limbs was noticed soon after birth. The condition had remained stationary, except in so far as the child had grown. The right side of the face, right half of tongue, right thigh, and right leg, were decidedly larger than the corresponding parts on the left side. The left forearm was decidedly, and the left arm was slightly larger than the right forearm and arm respectively. Skiagrams of the forearms and legs showed that the bones on the enlarged side were slightly thicker than those of the smaller side (?). There was no increase in length. The left testicle was slightly larger than the right.

Double Optic Neuritis.—Dr. C. O. HAWTHORNE.—Boy, aged 9 years, the subject of double optic neuritis of moderate degree, without other evidence of intracranial or nervous disease, or of disease in the thoracic or abdominal viscera. Some degree of anæmia, but by no means extreme, and Wassermann reaction negative. Had been under observation in hospital for fourteen days, and except for some headache on first day had seemed quite well and happy. Acuity of vision $\frac{6}{60}$ each eye. The history was of occasional attacks of headache commencing eight months ago, sometimes very severe, and in recent weeks often accompanied by vomiting. No admitted violence to the skull or of illness other than the above. There was a history of "consumption" in the mother's family and of "fits" in that of the father; a sister had a "tubercular knee."

Ulceration of the Soft Palate.—MR. E. D. D. DAVIS.—A girl, aged 13 years, was sent to Charing Cross Hospital by Dr. J. D. Rolleston with the history that she had been admitted to the Grove Hospital for diphtheria on April the 14th. One week before admission to the Grove Hospital she complained of sore throat but was otherwise quite well. Cultures of the fauces showed no diphtheria bacilli and direct smears showed no Vincent's organisms.

When seen by Mr. Davis on May the 19th there was a large irregular ulcer involving the left half of the uvula, soft palate, and left pillar of the fauces, with considerable loss of the left half of the palate. The ulceration was atypical of lupus, tubercle, or syphilis, but Wassermann's reaction was positive. There was no other lesion of the nose, pharynx, larynx, or ear.

Postscript, July the 1st.—The ulceration healed with mercury and potassium iodide, and an injection of novo-arseno-benzol (3.75 mg.). A definite history of infection of both parents was obtained.

Lupus of the Hard Palate and Gums.—MR. E. D. D. DAVIS showed the case for comparison with the above. The lupus was of some months' duration and of unusual distribution.

Recurrent Jaundice.—DR. CAUTLEY (for Dr. P. HAMILL) showed a girl, aged 13 years, who had been under observation since November, 1914, suffering from recurrent attacks of jaundice. From then up to the present time she had not been entirely free from pigmentation. During the exacerbations bile was present in the urine, the fæces were clay-coloured, and the liver was enlarged. In the intervals her general health was good. Since January, 1916, the spleen had begun to enlarge, and had extended more than 1 in. below the costal margin. During an exacerbation the blood-count was found to be: Red blood cells, 3.78 million; white blood cells, 10,800 per cubic mm.; hæmoglobin, 70 per cent.; colour index, 0.9 per cent. Differential count: Polymorphonuclears, 52 per cent., total number 5620; lymphocytes, 45 per cent., total number 4860; eosinophiles, 1 per cent., total number 100; basophiles, 2 per cent., total number 220. Very slight anisocytosis and poikilocytosis, no nucleated red cells seen. Corpuscular fragility normal. Loewi's pancreatic reaction was negative.

Unilateral Enlargement of the Tongue.—DR. CAUTLEY (for Dr. P. HAMILL)—A girl, aged 5 years, was brought to hospital with unilateral enlargement of the tongue. It was with difficulty that the tongue could be retained in the mouth. The enlargement appeared to affect the right side only, and involved the floor of the mouth. The papillæ were enlarged but did not resemble the minute vesicles commonly seen in cases of lymphangiectasis. Skiagrams did not reveal any corresponding hemi-hypertrophy of the cranial bones.

Congenital Defect of the Left Ulna.—MR. PAUL BERNARD ROTH showed a girl, aged 9 years. The case was described in the *Lancet*, May the 23rd, 1914.

The Rational Treatment of Rickets, with Illustrative Case.—DR. ERIC PRITCHARD.—(*Vide* p. 295).

The Teeth in Rickets.—DR. J. LAWSON DICK.—(*Vide* p. 332).

Abstracts from Current Literature.

Tuberculosis.

The period of life at which tuberculous infection most frequently occurs (*Med. Record*, 1915, LXXXIX, p. 47).—**S. A. Knopf**, as the result of inquiries addressed to well-known specialists on tuberculosis, internists and pædiatrists, comes to the following conclusions: Tuberculous disease in childhood is comparatively rare compared with tuberculous infection, which is exceedingly common. Most cases of tuberculosis in the adult have their origin in an infection during infancy or childhood. The frequency of infection increases with the age of the child, and is also affected by the environment. Under one year the percentage ranges from 1 to 9, from the first to the third year from 9 to 50, from the third to the fifth year from 27 to 75, from the sixth to the tenth year from 34 to 75, and from the eleventh to the fifteenth year from 12 to 94. Primary infection is most frequently found in the lungs and lymph nodules. Prenatal infection is perhaps more frequent than statistics show. The age at which a tuberculous infection contracted in infancy or childhood becomes active is according to the majority of Knopf's correspondents at or shortly after 15 years, next between 18 and 30 years.

J. D. ROLLESTON.

Frequency of infection with tubercle bacillus in childhood (*Am. Journ. Dis. Child.*, 1915, IX, p. 478).—**B. S. Veeder** and **M. R. Johnston**.—A study of tuberculin tests in 1321 cases at St. Louis Children's Hospital showed that the percentage of positive reactions reached a maximum of 44 per cent. at the age period of 10–14 years, including cases with clinical tuberculosis. If the latter were excluded, the percentage with positive reactions at the 10–14 age period was only 36. These figures are much lower than the 90 per cent. figure for the incidence of infection in children by their fourteenth year which is based on the figures of Hamburger for Vienna. The intradermic test gave only a slight increase in the percentage of positive reactions over the percentage of positive cutaneous reactions.

J. D. ROLLESTON.

The problem of tuberculous infection in children (*Boston Med. and Surg. Journ.*, 1916, CLXXIV, p. 755).—**H. W. Dana**.—A large proportion of all children are infected with tuberculosis in childhood, and from this early infection most of the active tuberculosis of later life is believed to spring. In the examination of 801 school children, 40 per cent. of whom were examined more than once, 12.1 per cent. were found to have probable tuberculous infection. It is impossible, in most cases, to determine whether the process shown by the X ray is active or not.

J. D. ROLLESTON.

What constitutes tuberculosis in childhood? (*Boston Med. and Surg. Journ.*, 1915, CLXXIII, p. 654).—**J. L. Morse** distinguishes between tuberculous infection when the disease is not present and tuberculous disease. In the former case children react positively to von Pirquet's test, but this only denotes infection, not disease. D'Espine's sign shows that there is probably enlargement of the tracheo-bronchial glands but not that they are necessarily tuberculous. Enlarged glands do not mean they are diseased

unless fever, malaise and debility are present. When a positive D'Espine is associated with a positive von Pirquet test, the chances are that the enlarged tracheo-bronchial glands are tuberculous, especially if the sign persists. If D'Espine's sign is positive and von Pirquet's test negative, the condition is certainly not tuberculosis. The significance of an X-ray plate is the same as D'Espine's sign. A definite history of exposure to tuberculosis is in favour of this disease if signs of debility, etc., be present.

F. R. B. ATKINSON.

A clinical study of 228 children in relation to tuberculous exposure controlled by von Pirquet reaction (*Am. Journ. Dis. Child.*, 1915, x, p. 354).—J. B. Manning and H. J. Knott, contrary to the findings of Fishberg (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1914, xi, p. 419, and 1915, xii, p. 345), found that children living in a tuberculous milieu and those with no known contact with consumptives showed marked differences; the former reacting in a relation of two to one of the latter. The number of positive reactions in the entire series was 42.9 per cent. The number of children between 10 and 15 with positive reactions in a series in which the majority came from tuberculous homes was 58.1 per cent., far below the figures of Hamburger, 95 per cent., and von Pirquet, 93 per cent. The writers attribute these discrepancies to peculiarities of climate, housing and sanitation in different communities.

J. D. ROLLESTON.

On the prognostic significance of a positive von Pirquet's reaction in children from 0 to 2 years of age (*Hospitalstidende*, 1916, lix, p. 697).—G. E. Permin.—A positive reaction at this age without any other symptoms is by no means so grave a prognostic as is usually supposed (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1913, x, p. 548). It has most significance in the first year of life, much less in the second. Of 15 children with a positive reaction in the first year of life, 6 died between the first and fifth year, while of 31 with a positive reaction in the second year only 2 died, between the second and third year. Many children who have been exposed at home to severe and fatal phthisis remain in good health after a long period of observation.

J. D. ROLLESTON.

Oral tuberculosis (*Annals of Otology*, 1915, xxiv, p. 193).—T. E. Carmody reports seventeen cases in full, and abstracts in tabular form 534 cases, with 661 lesions, distributed as follows: Tonsil 48, pharynx 51, palate 97, upper jaw 48, lower jaw 36, cheeks 16, tongue 277, lips 48, location not specified 40. The paper, which is profusely illustrated with colour and other plates, forms a useful *résumé* of its subject and requires to be read in its entirety.

MACLEOD YEARSLEY.

Digestive troubles in tuberculous children (*Arch. de Méd. des Enf.*, 1916, xix, p. 29).—L. Jeanneret divides them into: (a) Slight: the complexion is bad, the tongue furred, the appetite irregular and capricious, and there is a desire for spiced and acid foods. (b) Moderate: stomachic symptoms, heaviness after food, pain one to two hours after food, sometimes vomiting. (c) Advanced: intestinal symptoms, ending in all the signs of chronic enteritis. Out of 110 cases examined, 35 per cent. showed anachlorhydria. Treatment: (1) Avoid alkalies; (2) diminish foods requiring gastric juice for their digestion; (3) give pepsin and HCl; (4) treat the enteritis secondarily; (5) treat the tuberculosis.

F. R. B. ATKINSON.

On curable tuberculosis of the nervous centres (*Riv. di Clin. Pediat.*, 1916, xiv, p. 181).—**G. Fiore** having published in 1911 a case of cerebral tuberculosis which resulted in cure (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1912, ix, p. 279) and in 1913 an article on tubercle of the nervous centres shown anatomically to be curable (*Ibid.*, 1913, x, p. 552), came to the conclusion that the axiom of the incurability of meningeal tubercle could no longer be maintained in an absolute manner. He now publishes another case of tuberculous meningitis in which recovery was complete. A child, aged 8 years, who had convulsions when 1 month old, whooping-cough at the age of 5 years, and scarlet fever at 6 years, was admitted to hospital with marked signs of lymphatism and tracheo-bronchial adenitis, von Pirquet's reaction being strongly positive. At the same time there was broncho-pneumonia which cleared up leaving only a patch of harsh breathing at the apex of the right lung. Shortly afterwards there occurred vomiting, headache, drowsiness, irregular fever, increased reflexes, persistent dermographia and marked cranial tympanicity on percussion. Lumbar puncture gave abundant clear fluid under pressure, with lymphocytosis and albumin, which produced tuberculosis in a guinea-pig. The patient continued in the same state for more than a month and then began to improve, regaining consciousness. The residual signs of the apical broncho-pneumonia disappeared. Lumbar puncture was practised eight times, the pressure diminishing *pari passu* with the amount of morphological elements and albumin. After five to six months recovery was complete.

VINCENT DICKINSON.

A study of 105 cases of tuberculous meningitis (*Am. Journ. Dis. Child.*, 1915, ix, p. 426).—**A. E. Meyers** analyses the cases admitted to the Boston Children's Hospital since 1910. Fifty-two were males, 53 females, the youngest was 5 months, the oldest 11 years. Measles was noted in the past history in 26 per cent., whooping-cough in 22 per cent., and pneumonia in 11 per cent. The largest percentage occurred during January, April, and July, and the lowest in September, October and November. The average duration of the disease was 17 days, the shortest 5 days, and the longest 43 days. Lumbar puncture was done in all cases once in every forty-eight hours, and 20 to 30 c.c. removed. In 80 per cent. the fluid was under pressure, in 11 per cent. the tension was normal, and in 9 per cent. it was not recorded. Tubercle bacilli were found in the centrifuged clot in 21.5 per cent. The average count was 198 cells to the cubic mm. The prevailing type of cell was the small mononuclear, in only three cases was the polymorph the prevailing type. A positive test for globulin was found in 50 per cent., in 7 per cent. it was negative, and in 43 per cent. it was not recorded; 63 per cent. showed a positive von Pirquet's reaction, and 25 per cent. a negative reaction. Frequently when the first or second test was negative, the third proved positive. In over 35 per cent. of the positive cases the patients were unconscious on admission. There was a definite history of exposure to tuberculosis in 25 per cent., and no known exposure in 73 per cent. Treatment consisted in large doses of hexamethylenamine and sodium benzoate, and inunctions of mercury. In no case was there any recovery or remission. The paper contains an analysis of the symptoms and signs presented by the 105 cases, and a list of the necropsy findings.

J. D. ROLLESTON.

Acute general tuberculosis, with left oculo-motor paralysis after measles (*Dub. Journ. Med. Science*, 1916, i, p. 174).—**J. Moore** records

this case in a girl, aged $4\frac{1}{2}$ years, who had always been healthy until she contracted measles about nine weeks before admission to hospital. On the day before admission complete ptosis of the left eyelid developed suddenly and slight slurring of the speech had been noticed. The evening temperature registered 102° F. On admission the axillary temperature was only 96° F., the pulse-rate was 86, and the respirations were 28 per minute. The child was quite conscious and very intelligent. She answered questions more by gestures than by speech. Her tongue was thickly coated. The most striking signs of illness were in connection with the left eye. There was complete paralysis of the left third nerve. Absolute ptosis, with dilatation of the left pupil, inability to turn the eye inwards or upwards, slight external strabismus, and loss of power of accommodation and of contraction on exposure to light. The fourth and sixth nerves seemed to be unimpaired in power and function. Examination of the left fundus showed that the disc was somewhat pale; no congestion detected; isolated patches of choroiditis noted; choroid very thin, as was also the case on the right side. The child died eleven days after admission, and the post-mortem findings were as follows: Disseminated tuberculosis of both lungs and also of the mediastinal and peribronchial glands. Very profuse tuberculosis of the pericardium, diaphragm, liver, kidneys, spleen and peritoneum. Extensive tubercular basal meningitis. No involvement of the nucleus or the intracranial course of either third nerve was observed on hardening and examining sections.

J. ALLAN.

Disseminated miliary tuberculosis (*'Arch. of Ped.,'* 1915, xxxii, p. 887).—**W. P. Northrup**.—The patient was an infant, aged 15 months, whose mother and brother were tuberculous. The disease began at 9 months with wasting and cough. The physical signs in the chest were indefinite and no tubercle bacilli were found in the sputum or fæces, but the X rays showed the presence of miliary tubercles. The skin of the trunk, especially the back, face and forearm, showed papulo-pustular umbilicated lesions (necrotic tuberculides). Some pea-sized nodules were found in the subcutaneous tissue in the left forearm, in the arm and in each calf. One nodule was removed and proved to be tuberculous. The autopsy showed widespread miliary tuberculosis in the lung and bronchial glands, tuberculous ulcers in the intestines, and tuberculous meningitis.

J. D. ROLLESTON.

Rapidly advancing pulmonary tuberculosis (*'Arch. of Ped.,'* 1915, xxxii, p. 735).—**Fairfax Hall**.—In the normal lung the X ray exposes branchings from the hilus, representing bronchi, vessels, and glands. In the tuberculous lung there is enlargement and diffusion of these shadows representing enlarged peribronchial glands. Miliary tubercles cast small spotted shadows, larger areas, more diffuse shadows, and cavities are seen as light areas in the midst of consolidated lung. A case of a girl, aged 2 years, in whom pneumonia and otorrhœa preceded febrile disturbance and cough, is described. At first only moist *râles* were heard at the bases, but shadows of enlarged glands were shown at both roots, and the von Pirquet test was strongly positive. Gradual extension of the X-ray shadows was noted on the forty-fourth day of the illness. Signs of a cavity at the right base were detected by the radiologist, and infiltration of the lung by the clinician. Ten days later there was marked dullness at the right base with bronchial breathing below the angle of the scapula. The von Pirquet reaction was now much less marked. Loss of weight was now first noted.

On the sixtieth day of the disease tubercle bacilli were noted in the sputum and faeces. On the sixty-fifth day death supervened. Miliary tubercles were found in both lungs, with cavities, small in the left but large in the right, in both lower lobes. By the X rays the early diagnosis of the disease was ensured.

CHRISTOPHER ROLLESTON.

Ulcerative pulmonary tuberculosis in infants (*Le Progrès Méd.*, 1916, p. 34).—E. Aine makes this the subject of his *Thèse de Paris*. It is not exceptional, he says, to meet with more or less extensive ulcerative lesions of the lungs at autopsies of infants dead of tuberculosis, analogous to those which characterise the usual evolution of pulmonary tubercle in the adult. Anatomically they occur in four different forms: (1) The simple vomica, usually single and apparently representing the primary nodule of infection. It is seen most often in very young infants, from 3 to 6 months, and has a scanty symptomatology usually extra-pulmonary. (2) In the majority of cases the vomica is hollowed out in the middle of a patch of caseous pneumonia whose clinical evolution is usually subacute. It is usually large and multilocular. (3) The formation of vomicae by caseous broncho-pneumonia is a variant of the above process, differing from it in the fact that the vomicae are small and multiple; its evolution is generally more rapid. (4) When in either of the preceding types there is marked fibrous reaction, disseminated vomicae are found occupying one lobe or even an entire lung, in the midst of dense fibrous tissue with dilated bronchi. Clinically these forms give rise to few symptoms, physical signs are inconsistent and late, dullness persistent at the apex being alone excepted they are fallacious. Functional signs have greater value, especially hæmoptysis, which, however, is rare. Radiography is of special help. In conformity with current opinion, the author does not consider a congenital origin of these lesions frequent and has been unable to find any undoubted case of contagion *in utero*. The surroundings of the patients favoured the explanation of contagion by inhalation. From the social point of view, the types of pulmonary tuberculosis studied by Aine have a special interest from their relative frequency and their gravity. It is important to bear in mind that the infants attacked are the agents of infection, as proved by examination of their sputum. For this reason all infants who cough should be subjected to frequent and systematic examination even in the absence of an intra-dermal or cuti-reaction.

VINCENT DICKINSON.

Lymphogranuloma malignum (*La Pédiatrie*, 1915, xxiii, p. 481).—G. Caroina describes two cases of this disease (Hodgkin-Sternberg). In both typical forms of Much's granules were found but no tubercle bacilli. In the first case an intraperitoneal injection in a guinea-pig was made. An autopsy after three months showed tuberculosis of the peritoneum and abdominal glands with acute secondary miliary infection in the liver, spleen and lungs. In smears from the grey granulations in the latter, tubercle bacilli of granular form were found. From another guinea-pig, inoculated with the splenic pulp of the first animal, a bacillus resembling the human tubercle bacillus was cultivated.

VINCENT DICKINSON.

Treatment of tuberculous sinuses (*Glasg. Med. Journ.*, 1916, i, p. 107).—C. Bennett recommends packing tuberculous sinuses with gauze soaked in a solution containing 5 gr. of menthol in a drachm of methylated spirit. After application the dressing need not be changed for three days.

No undesirable local or general after-effects have been noticed. The local anæsthetic action prevents post-operative pain, the granulations of the sinuses remain of a healthy type, discharge is rapidly reduced to a minimum, and the period of healing is noticeably shortened.

J. ALLAN.

Reviews.

TRAUMATIC PNEUMONIA AND TRAUMATIC TUBERCULOSIS. By F. PARKES WEBER, M.D., F.R.C.P.Lond. Reprinted, with additions, from articles in 'The Clinical Journal,' London, July, 1916. Pp. 63, $7\frac{1}{4} \times 4\frac{1}{4}$. London: Adlard & Son. Price 6d.

DR. PARKES WEBER has performed a very useful piece of work in bringing together, round a nucleus of his own observations, many of the references in medical literature to the subject of his reprinted papers. Traumatic or contusional pneumonia, and traumatic tuberculosis, are of particular interest in connection with the difficult subject of compensation for accidents and accidents in general. Both are diseases of adults far more than of the very young, although the author quotes a number of examples of each in children. The term "contusional pneumonia" was introduced by Litten in 1881, though the relation of pneumonia to injuries of the chest had been noted long before by Morgagni; the pneumonia may follow the injury at an interval of from two, four, six, or ten hours to many days, perhaps even a fortnight. As regards traumatic tuberculosis, meaning thereby cases due to injuries by which tubercle bacilli could not have been introduced from without, Dr. Parkes Weber divides the cases into three clinical groups. First he places those in which a decided traumatism of some kind is followed by acute generalised miliary tuberculosis with death in perhaps a month, or by acute localised metastatic tuberculosis in some uninjured part of the body; such cases may follow operations or manipulations of tuberculous joints, for example. Second he counts cases in which signs of pulmonary tuberculosis follow, or are first noticed, after a supposed injury to the lungs; such instances perhaps explain how it is that athletes so often succumb unexpectedly to phthisis. Third, he discusses instances in which injury to bones, joints, or parts of the body other than the lungs, is followed by signs of tuberculosis more or less localised to the region of the trauma. Generally this is to be regarded as the result of the disturbance of an unrecognised tuberculous focus already in existence but latent or quiescent. Dr. Parkes Weber's well-documented paper will be read with interest by all who are interested in the important subjects with which it deals.

A. J. J.-B.

ANATOMÍA DE LOS CONDUCTOS BILIARES Y DE LA ARTERIA CÍSTICA. By DR. P. BELOU, Titular Professor of Descriptive Anatomy at the Buenos Aires Faculty of Medical Sciences. $11\frac{1}{2}$ in. $\times$ 8 in. Pp. 302, with 102 figures and 22 plates, many of them in colours. Buenos Aires: Oceana Press, 1915.

IN this handsome and well printed monograph Professor Belou remarks that our knowledge of the extrahepatic bile-ducts has advanced very considerably during the last thirty years, in consequence of the progress made by operative surgery. His own work is based on the dissection of scores of adults, infants, and lower animals. More than two-thirds of the book

consist of elaborate descriptions and statistical figures relating to the cystic and hepatic ducts; the author distinguishes five segments of the hepatic duct, namely the initial, the epiploic, the retroduodenal, the retropancreatic, and the parietal or parietoduodenal. Then follow short chapters on the blood supply and anatomical anomalies of the bile-ducts, and on the general characters and disposition of the bile-ducts in the lower animals. There are thirty pages, with thirty figures, on the anatomy of the cystic artery, a subject to which Gray's *Anatomy* devotes four lines. The volume ends with a bibliography containing over 250 references to the literature; the author appears not to be acquainted with Dr. H. D. Rolleston's invaluable volume on the liver, gall-bladder, and bile-ducts. Professor Belou's monograph is a book for anatomists in the first instance; it also contains, scattered throughout its pages, a deal of information that should be of service to the surgeon or to the pathologist. But the volume has no general index, alas! and so it is unlikely that the stores of information it contains will ever be unlocked in this country. A special word of praise may be given to the water-colour sketches and pencil drawings of Professor Belou's artist, Mr. A. Bouvet.

A. J. J.-B.

A CODE OF RULES FOR THE PREVENTION OF INFECTIOUS DISEASES IN SCHOOLS. Issued by the Medical Officers of Schools Association. Seventh edition. London: J. & A. Churchill, 1915. Price 1s.net.

THIS useful pamphlet begins with an introduction on general hygiene, but here several precautions are omitted. No instructions are issued on the proper care of earth closets, and no details are given as to the adequate protection of well water. The milk and food supply receive but scanty attention from public school authorities, and the same may be said of the dairy. Few, if any, schools have a dairy of their own. No instructions are given concerning the examinations of dairy employees, cooks, and other domestic servants; but surely in these expensive establishments serological and other tests should be provided to prevent infection by Eberth's bacillus, the tubercle bacillus, and the spirochæte of Schaudinn. With regard to the actual rules of isolation considerable alteration has been made since the last edition. Two negative swabs are considered sufficient in diphtheria, but nasal as well as oral cultures are advised. In scarlet fever it is pleasing to note that no importance is attached to desquamation. Very pertinent remarks are made on the importance of microscopical examination of ring-worm. A most useful appendix on disinfecting and disinfectants is contributed by Dr. Houston.

C. R.

HANDBOOK FOR WIVES AND MOTHERS IN INDIA. By MILDRED E. STALEY. London: W. Thatcher & Co. Price 5s. net.

THE 'Handbook for Wives and Mothers in India,' by Dr. Mildred E. Staley, has reached a second edition. It is written by one who combines a sound knowledge of medicine and hygiene with a full understanding of the special circumstances of life in India. We can especially commend the part of the book which deals with the management of infants and children in health and disease. The author has clearly studied closely the most recent publications on the subject, and of these she has made a very judicious use. On p. 242 the statement is made—and we doubt its truth—that if citrated whole milk is used in feeding, "the infant will need *only half the amount of plain milk* to that usually given in each feed of diluted milk." The italics are the author's.

H. C. C.